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EMERGENCY

What is New with Taurine?

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The cardiac disease dilated cardiomyopathy (DCM) has been genetically linked to certain dog breeds and can also be seen post doxorubicin administration. At Angell Animal Medical Center (AAMC), we have been diagnosing an increasing number of dogs with DCM and have been wondering why this is the case.

In 1987, Pion et al.'s article "Myocardial failure in cats associated with low plasma taurine: a reversible cardiomyopathy" was published in *Science*. The authors reported the discovery that taurine deficiency in cats can cause DCM.[1] Subsequent articles by many of the same authors investigated the supplementation of taurine in these cats.[2] Cats are obligate carnivores and cannot make taurine, so the amino acid is considered essential. Unlike cats, dogs can make taurine from other dietary amino acids and therefore don't have to rely on diet.

Especially noteworthy is that the breeds we have recently been diagnosing with DCM are not the breeds known to have a genetic predisposition (dobermans, boxers, and American cocker spaniels). We are seeing DCM in "atypical" breeds like Boston terriers, etc., and the commonality among these patients is that they are being fed a grain-free or homemade diet. Some recent cases diagnosed at AAMC include a dog being fed a vegetarian diet his whole life that was high in lentils and a dog on a strictly vegan diet. Both dogs developed taurine

INTERNAL MEDICINE

Atypical Addison's Disease

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Canine glucocorticoid deficient hypoadrenocorticism or "atypical" Addison's disease is being identified in our canine population with greater frequency. The term describes dogs with adrenal glands that still produce mineralocorticoids but lack sufficient glucocorticoid production. Atypical Addison's disease used to be considered a much rarer condition than the classical Addisonian but now is identified in up to 30-45% of dogs diagnosed with hypoadrenocorticism. Both types of Addison's disease still occur with greater frequency in younger dogs, with the average age of diagnosis about four to five years of age. Female dogs account for about 70% of the cases, and there is a heritable component in standard poodles, bearded collies, Portuguese water dogs, and the Nova Scotia duck tolling retriever.

Atypical Addisonian dogs usually have more subtle clinical signs and may be more difficult to diagnose. Usually these dogs do not present in a crisis, nor are they severely dehydrated or bradycardic or in hypovolemic shock, as they still have mineralocorticoid function. Their signs are glucocorticoid dependent and subtler. Clinical signs vary, but may include vomiting, diarrhea, anorexia, lethargy, or weight loss. The most typical history we see is a young dog with chronic intermittent histories of gastrointestinal issues. They commonly have episodes or signs that "wax and wane." Some of these

EMERGENCY

FIGURE 1

deficiency and DCM. The grain-free diets have become very popular in recent years, so perhaps it is the formulations that are causing the issue. An article by Ko et al. tested whether or not rice bran or other fibers, including beet pulp, affected bile acid excretion. They did not find evidence that rice bran was causing low taurine levels in large dogs being fed lamb and rice diets; however, they reported that beet pulp may have lowered the whole body taurine in dogs by increasing excretion of fecal bile acids and decreasing protein digestibility. This would decrease the availability of sulfur amino acids, which are precursors for taurine.[3] In addition, we have diagnosed five cats within a six-month period with DCM that had been eating grain-free/boutique diets and had confirmed low taurine levels.

In a recent online article,[4] Dr. Lisa Freeman, a nutritionist at Tufts University's Cummings School of Veterinary Medicine, said in regard to DCM in non-taurine-deficient dogs, "What seems to be consistent is that it does appear to be more likely to occur in dogs eating boutique, grain-free, or exotic ingredients diets." Exotic ingredients include novel proteins (venison, buffalo, kangaroo) and legumes like lentils, chickpeas, etc. Dr. Freeman calls them Boutique Exotic Ingredients (BEG), and more information can be found in her article at the following link: http://vetnutrition.tufts.edu/2018/06/a-broken -heart-risk-of-heart-disease-in-boutique-or-grain- free-diets-and-exotic-ingredients.

The best way to determine if a pet has taurine deficiency is to run a whole blood taurine level. Plasma levels can reflect acute changes; therefore, whole blood levels will be more accurate in

determining the whole-body taurine level. For DCM in cats, the recommended supplementation taurine dose is 250 mg PO SID to BID. For DCM in dogs under 25 kg, the dose is 500-1000 mg taurine PO BID to TID. For dogs over 25 kg, 1-2 grams taurine PO BID or TID is recommended.[5]

A whole-blood taurine level costs about $200 at Wisconsin Veterinary Diagnostic Lab. Samples should be shipped in heparin (green top tube), and the test results take about five to seven days to return. The reference range for whole-blood normal taurine levels in dogs at this lab is 200-350 nmol/mL, with a critical level of <150 nmol/mL.

Supplemental taurine is easy to obtain at local drug stores or nutrition stores. It comes in many different formulations, including chewable tablets, capsules (250 mg, 500 mg, 1000 mg), liquid, and powder. Amazon.com sells a 100-count bottle of 1000 mg taurine capsules for approximately $20.

Currently, there are no published studies confirming a link between boutique/grain-free diets and DCM, but several studies are underway. Dr. Joshua Stern, a cardiologist from UC Davis, is conducting research on golden retrievers with DCM and taurine deficiency.

Not all pets diagnosed with DCM while being fed a grain-free or limited-ingredient diet respond to taurine supplementation, but some certainly do. The AAMC Cardiology service recommends starting taurine supplementation and switching to a more balanced diet if the patient is not on their current special diet for a medical reason. In addition, a recheck of the whole-blood taurine

level and echocardiogram should be performed three months after supplementation is started.

This article is not to suggest that all boutique diets will cause harm. However, feeding a diet developed by a company using evidence-based nutritional research that can guarantee the contents of its product is recommended. Many small, specialty boutique companies are unable to make these claims. Reporting cases of DCM suspected to be a result of diet to the FDA will help further elucidate the problem. At this time, it is not certain why these dogs have DCM; most cases are not taurine deficient, and not all are being fed a grain-free diet.

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3 Ko et al. Dietary beet pulp decreases taurine status in dogs fed low protein diet. *J Anim Sci Technol* 58(29), 2016.

4 A broken heart: risk of heart disease in boutique or grain-free diets and exotic ingredients. Online article at vetnutrition.tufts.edu by Dr. Lisa Freeman, June 4, 2018.

5 Plumb's veterinary drug handbook, 7th Edition. Ames, IA: Wiley-Blackwell, 2011, 958-959.

FIGURE 2
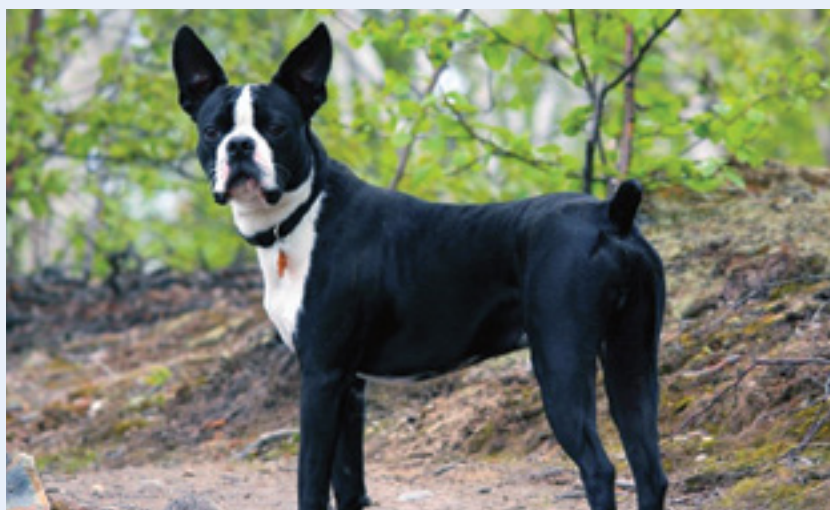
INTERNAL MEDICINE

FIGURE 1

dogs have been sent in for gastrointestinal (GI) workups, including the recommendation for endoscopic biopsies. Atypical Addison's should be ruled out before endoscopy.

Addison's disease has often been called the "great imposter," as it can mimic many other diseases and is often difficult to initially diagnose. Since dogs with atypical Addison's disease still have mineralocorticoid function, they lack the typical electrolyte abnormalities commonly associated with hypoadrenocorticism. Their sodium and potassium values are often normal, so other blood abnormalities need to be noted to guide in the diagnosis. A lack of a stress leukogram in an ill animal is commonly seen in animals with a cortisol deficiency.

Subtler lab abnormality signs include a mild to moderate hypoalbuminemia and hypocholesterolemia, once again pointing to GI disease, but decreased cortisol levels can also be the problem. When a young dog presents for only hypercalcemia or eosinophilia, atypical Addison's needs to be considered. Other dogs, especially toy breeds, may present just with hypoglycemia. A dog could have only one of these lab abnormalities or multiple. It is important to rule atypical Addison's out before getting into more extensive workups in all these conditions.

An excellent and cost-efficient way to rule out atypical Addison's disease is by running a basal cortisol level. If your patient's basal cortisol level is over 2 mcg/dl, the dog is unlikely to have Addison's disease. Values below 2 mcg/dl don't diagnose the disease; they just mean that the dog could have Addison's disease and an ACTH stim should be run. Even if the basal cortisol level is at the lowest level detectable for your lab, an ACTH stim needs to be done to diagnose the condition, as some dogs that have very low basal cortisol levels still will have cortisols that stimulate to acceptable levels.

Dogs with Addison's disease usually have ACTH stims with the pre- and post-cortisol under 1 mcg/dl, but if both values are under 2 mcg/dl, it is diagnostic for Addison's disease.

Occasionally, in dogs that are tested early in disease progression, the cortisol concentrations may be slightly higher. "Flat-line" cortisol responses can be seen when a pre-ACTH cortisol level might be 3 mcg/dl and a post-ACTH cortisol might be 3.3 mcg/dl. Retesting these dogs in four to six weeks might show more definitive results.

Another factor that could confound a diagnosis is if a dog has received prednisone before undergoing either a basal cortisol level or an ACTH stim. Giving prednisone prior to doing a cortisol level will falsely increase the measured cortisol, possibly giving a false-negative result.

The treatment of glucocorticoid-deficient Addison's disease involves using the lowest effective dose of prednisone that controls the dog's clinical signs and minimizes any long-term prednisone side effects. Typically, after initially establishing a diagnosis, higher doses of prednisone may be used (up to 1 mg/kg/day), but these doses may be rapidly decreased to a maintenance dose closer to 0.1-0.2 mg/kg/day. All dogs are different, and some dogs need less than 0.1 mg/kg/day or only need it every other day. As with typical Addisonian dogs, in stressful situations, the maintenance dose of prednisone is usually doubled.

Sometimes dogs receiving long-term prednisone for the treatment of their disease begin to show side effects of long-term prednisone administration (the most common are polyuria/polydipsia/primary polydipsia [PU/PD/PP]) even at fairly low prednisone doses. In these dogs, changing prednisone to methylprednisolone may significantly improve their clinical signs.

The key in treating all atypical Addisonian dogs is to give them the smallest amount of prednisone or methylprednisolone to control their clinical signs and minimize any potential side effects of the corticosteroid. If doses of prednisone significantly higher than 0.2 mg/kg/day are needed to control a dog's clinical signs, then it is possible that the patient may have another disease that the prednisone is treating. The atypical Addisonian dog that had chronic intermittent GI signs and requires higher doses of prednisone may also have inflammatory bowel disease (IBD), and the higher doses are actually treating the IBD. Establishing the diagnosis of IBD would allow you to treat the IBD with different modalities, allowing for a smaller dose of prednisone and thus fewer side effects.

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Dogs with atypical Addison's disease typically live excellent, quality lives, and the disease does not affect the dogs' life expectancy. Dogs should be evaluated at least twice a year, and electrolytes should be monitored. It is rare that a dog with glucocorticoid-defcient Addison's will develop classical Addison's, with electrolyte abnormalities, but it has been reported.

Some have suggested that it is safer to prophylactically give these dogs DOCP to reduce the chance of an Addisonian crisis that could develop if a dog becomes mineralocorticoid defcient as well. Most veterinarians no longer recommend this due to expense and the unlikeliness that their disease will progress. Routine monitoring with physical examinations, blood work, and client education is usually suffcient.

Remembering atypical Addison's as a possible cause of many subtle signs or specific lab abnormalities can save you and your clients extensive diagnostic workups and signifcantly improve the quality of life of your patients.
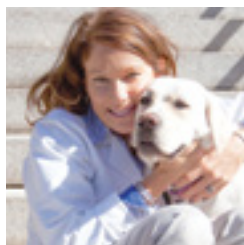
Liver Disease and the Intestinal Microbiome

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The gut microbiome has emerged once again in the treatment of systemic disease, this time as it relates to disease of the liver. Various types of liver disease in people have been associated with alterations in the intestinal microbiota, so it may be fitting at this time to investigate the role of the microbiome as it relates to diseases of the liver in animals, as a factor related to primary liver disease as well as to bile acid metabolism.

The microbiome is a population of microorganisms that live in an established environment and include bacteria, fungi, viruses, and parasites. The intestinal microbiome includes one of the densest microbial populations on the planet—100 trillion microorganisms per average human. The microbiome is a factor in the maintenance of mucosal integrity and normal permeability of the intestinal mucosa.

Intestinal dysbiosis is a shift in the balance of gut microbes—both in types of organisms and diversity—adversely affecting the health of the host. Dysbiosis has been shown to be related to many diseases in human medicine, including primary intestinal disease (e.g., ulcerative colitis), auto immune disease (e.g., multiple sclerosis), autism, metabolic syndrome/obesity, and more recently, diseases of the liver and bile acid metabolism.

Blood flow to the liver is composed of flow from the hepatic artery (which delivers oxygenated blood to the liver) and from the portal vein, which delivers deoxygenated blood from the intestinal tract and accounts for 75% of the blood flow to the liver. It makes sense, therefore, that abnormal intestinal microflora would directly affect the liver, as bacteria, bacterial byproducts, cytokines, toxins, and metabolites are all delivered directly to the liver during first-pass metabolism.

Various types of liver diseases in humans have been discovered to be related to intestinal dysbiosis, including primary sclerosing cholangitis (PSC), fatty liver disease (NAFLD), and cirrhosis, as well as alcoholic liver disease (ALD).

FIGURE 1

↘ In different hepatic diseases, an impaired intestinal microbiota has been defined at left. Increased gut permeability results in increased translocation of lipopolysaccharides (LPS) and inflammatory responses and mediators such as TNF.
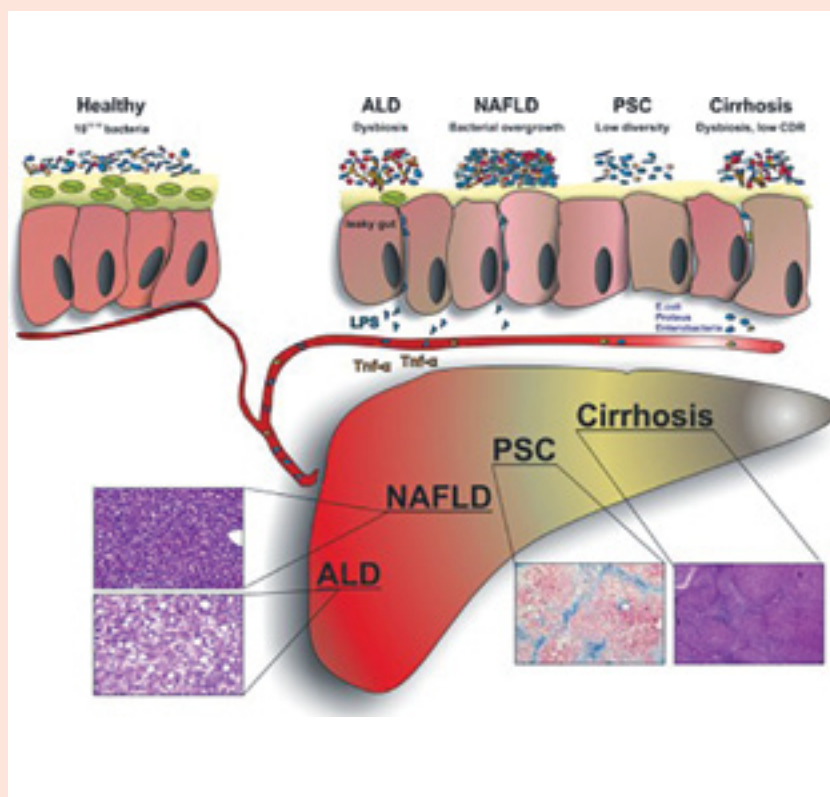

Diagram obtained/adapted from Tilg H, Grander C, Moschen AR. Clinical Liver Disease 2016 Nov; 8(5): 123-126.

In NAFLD, patients often are identified with alterations in intestinal permeability, bacterial overgrowth of pathogenic species, and increased circulating levels of endotoxin and TNF α. The "microbiome signature" in these patients appears to regulate liver and tissue fat storage and is potentially a contributing factor to the pathogenesis of disease progression in these liver patients. PSC, also associated with a "microbiome signature," is commonly associated with people who suffer from inflammatory bowel disease. In these patients, there has been shown to be reduced bacterial diversity compared with control patients, as well as an increase in a species of bacteria, *Veillonella*. Furthermore, when nude mice undergo fecal transplantation with material from PSC patients, these animals actually show biochemical features consistent with PSC. Furthermore, *Veillonella* (part of the phylum *Firmicutes*) has

also been associated with other chronic inflammatory disorders, including fibrotic conditions. In cirrhotic patients, progressive abnormalities in the gut microbiota accompany disease decompensation associated with increased translocation of bacteria and increased circulating levels of bacterial DNA.

Based on the findings above, there is likely a role for prebiotics and probiotics, as well as fecal microbiota transplantation (FMT), in the treatments of these human diseases and potentially for the treatment of our veterinary patients. In addition, conditions associated with liver disease, such as hepatic encephalopathy, might also be associated with dysbiosis.

To complicate the liver-dysbiosis relationship, once changes in liver integrity have occurred, the dysbiotic state can become progressively more deranged due to altered bile acid synthesis and metabolism.

Primary bile acids are synthesized by the liver and secreted into the intestinal lumen. Primary bile acids in dogs include cholic and chenodeoxycholic acid and are produced in the liver and conjugated to taurine or glycine to form bile salts before secretion into bile to assist in lipid digestion in the small intestine. Approximately 95% of secreted bile salts are reabsorbed from the small intestine via the enterohepatic circulation, with the other 5% reaching the colon. Enzymatic reactions catalyzed by gut bacteria lead to the production of secondary bile acids, which include deoxycholic acid, ursodeoxycholic acid, and lithocholic acid.

Studies have shown that in dogs with chronic enteropathy and dysbiosis, certain secondary bile acid concentrations are decreased. Abnormal fecal flora can result in an alteration in the process of hydrolysis of bile salts and conversion of primary bile acids to secondary bile acids. Because secondary bile acids can prevent germination of certain nefarious bacterial species in the colon, a reduction in these can result in bacterial overgrowth that results in a dysbiotic state. Conversely, an increase in primary bile acid concentrations can actually promote the germination of unsavory species.

Furthermore, as we now know, the liver can be profoundly affected by intestinal dysbiosis and diminished intestinal microbial diversity. Not only does abnormal fecal flora result in reduced diversity and/or increases in pathogenic species and delivery of bacterial byproducts, metabolites, and toxins to the liver, but the affected liver then engages in bile acid synthesis, whose abnormal metabolism perpetuates the dysbiotic state.

FMT in humans has shown a reversal of fecal bile acid profiles as it relates to the treatment of *clostridium difficile*. The working theory is that the correction of bile acid metabolism may be a mechanism by which fecal transplantation results in a cure in these patients.

Conclusion

The intestinal microbiome is progressively becoming more and more relevant as it relates to the pathophysiology and treatment of diseases involving many body systems. Manipulation of the microbiota via FMT or the use of prebiotics or probiotics needs further investigation as to how these therapies relate to liver inflammation, fibrosis, fat accumulation, and other advanced liver diseases.

At Angell I have been performing fecal transplants in animals with GI disease for many years. Presently we are submitting Fecal Dysbiosis Indices on potential FMT patients with gastrointestinal disease (Texas A&M GI lab), and we are performing FMT via enema (cats) or capsules (dogs). As we further investigate the role of the microflora as it pertains to systemic disease, the liver is next on our list of organ systems we will consider.

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Surgical Management of Canine Glaucoma: Show Me the Numbers

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Glaucoma is one of the most common eye diseases and causes of blindness of dogs. Although there are numerous medical therapies for glaucoma, there is no de facto cure, and many cases will eventually develop a breakthrough elevation in intraocular pressure (IOP), resulting in pain and vision loss.

When medical management of glaucoma fails, there are vision-saving surgical options available. The parameters for making a surgical decision are quite nuanced, depending on the individual patient, client, and even the attending ophthalmologist. Since the risks of surgery can include IOP exacerbation and blindness, the risks and benefits of any surgical procedure must be carefully considered. There are only a handful of articles reporting actual success rates of surgical management of glaucoma. This article presents reported objective success rates to better inform subjective decision-making parameters.

Just as with medical management, there are two general categories of surgical therapy—those that increase outflow of aqueous humor (drainage or shunting procedures) and those that halt the production of aqueous humor (via destruction of the ciliary body or cycloablation). In veterinary medicine, cyclodestructive procedures tend to enjoy greater success than shunting procedures, although often the two are combined. Over the past 20 years, there has been an evolution of surgical glaucoma management, starting with transscleral application of first liquid nitrogen and then diode laser energy to the ciliary body, continuing with endoscopic lasering from within the eye, and most recently coming full circle to transscleral application of MicroPulse® laser energy. The lines are blurred between these "past," "present," and "future" therapies, with no single procedure standing out definitively as a gold standard.

THE PAST?

Transscleral cyclocryotherapy, or the application of a liquid nitrogen-cooled contact probe to the sclera overlying the ciliary body, was initially reported for surgical control of glaucoma. This was then largely and quickly replaced with diode laser transscleral cyclophotocoagulation (TSCP). In this procedure, diode laser energy is applied with a contact probe positioned external to the sclera, 3-4 mm posterior to the limbus, presumptively overlying the location of the ciliary body (Figure 1).

TSCP can be combined with aqueous humor drainage devices. The Ahmed valve gonioimplant is the most popular of such devices (Figures 2-4). It consists of a footplate that is placed between the sclera and conjunctiva and is sutured to the sclera, with tubing that is inserted into the anterior chamber. Aqueous humor thus flows out of the tubing into the footplate area, where it is absorbed by the conjunctiva. Short-term failure can occur due to occlusive fibrin formation in and around the tubing. Long term, this and scar tissue occluding the footplate absorption area, or extrusion of the implant, can also cause failure.

↘ Transscleral cyclophotocoagulation for glaucoma (in a rabbit), with the diode laser unit inset. The globe is stabilized with forceps at the limbus and laser energy applied in a transscleral fashion, posterior to the limbus overlying the presumptive location of the ciliary body.

↘ Ahmed valve gonioimplant (footplate with suture holes to the right, tubing to the left).
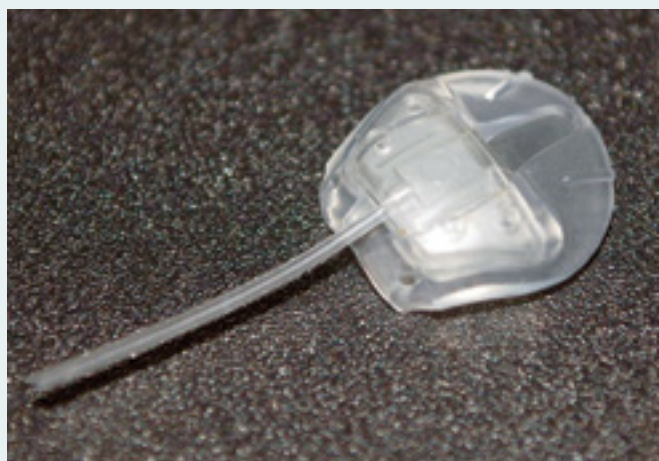
OPHTHALMOLOGY

FIGURE 3

↘ Ahmed valve gonioimplant in place in the left eye of a dog. Note the tubing entering the eye at the limbus at the one o'clock position.
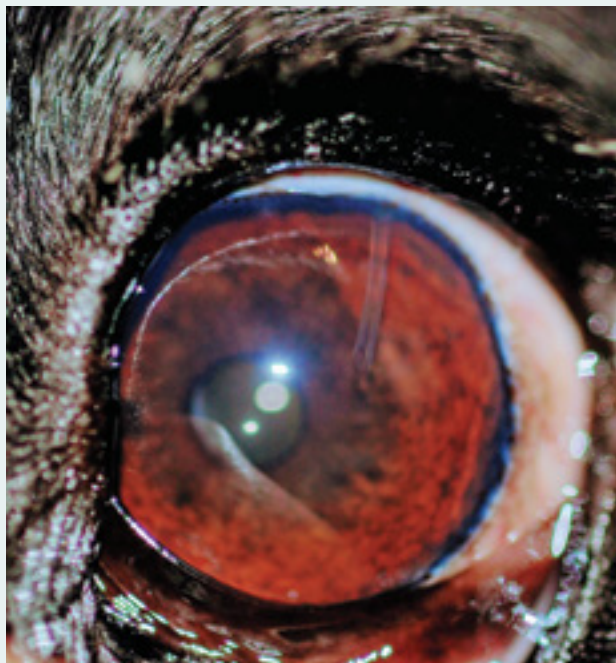
FIGURE 4

↘ Two Ahmed valve gonioimplants in place in the right eye of a dog. Note the tubing entering the eye at the limbus at the 7 and 11 o'clock positions. This dog maintained intraocular pressure and vision for a year following TSCP and the first Ahmed valve placement, but a second valve was then placed for recurrent glaucoma.
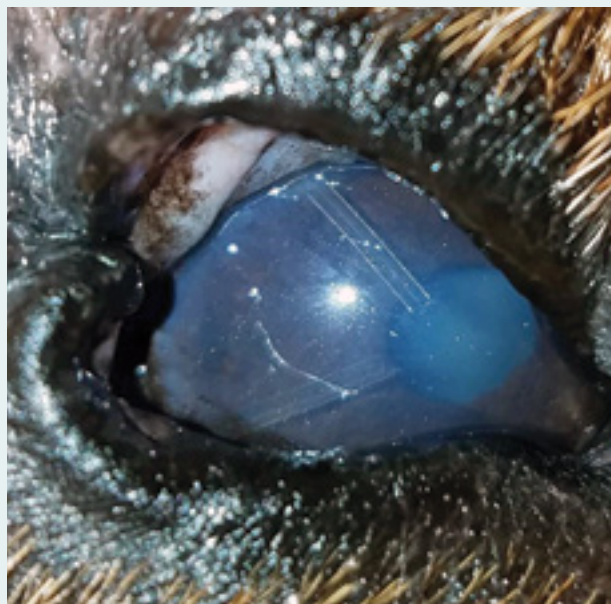
Table 1 presents a summary of the one-year outcome for IOP control and vision in six reports on the use of TSCP alone or in combination with Ahmed valve placement. Control of IOP for one year was reported in 51-92% of patients, whereas vision was present at one year in 41-89% of patients (although all but one study was in the 40-60% range for vision). Combining all these studies into one patient population results in one-year IOP control in 132/197 (67%) and one-year vision in 56/105 (53%).

TABLE 1

↘ Summary of six peer-reviewed studies on diode laser transscleral cyclophotocoagulation (TSCP), with or without Ahmed valve placement, for control of glaucoma in dogs. ICLE, Intracapsular Lens Extraction

YEAR	AUTHOR	TECHNIQUE	DISEASE	1-YEAR IOP CONTROL	1-YEAR VISION
1997	Cook *et al*	TSCP	Primary Glaucoma	45/88 (51%)	10/19 (53%)
1999	Bentley *et al*	Cryo or TSCP*	Glaucoma Not Defined	14/19 (74%)	11/19 (58%)
2001	Hardman & Stanley	TSCP	Primary Glaucoma	22/24 (92%)	7/14 (50%)
2003	O'Reilly	TSCP	Glaucoma Post-ICLE	12/15 (80%)	8/15 (53%)
2005	Sapienza & van der Woerdt	TSCP + Ahmed	Primary Glaucoma	39/51 (76%)	12/29 (41%)
2011	Westermeyer *et al*	TSCP + Ahmed	Primary Glaucoma	"Mean IOP control 722 days"	8/9 (89%)
Total of all reports				**132/197 (67%)**	**56/105 (53%)**

*The 1999 study includes cases treated with liquid nitrogen cyclocryotherapy instead of laser.

OPHTHALMOLOGY

THE PRESENT?

Building on this technique, diode laser endoscopic cyclophotocoagulation (ECP) is a more recent advance in glaucoma therapy. By inserting an endoscope directly into the eye, typically via a limbal incision, the ciliary processes of the ciliary body can be directly visualized (Figure 5) and thus laser energy can be applied more directly than is possible via the transscleral approach. This allows lower energy settings to be used, potentially reducing complications such as inflammation and hemorrhage and thus improving the potential for success. Unfortunately, the lens precludes visualization and treatment of the entire ciliary process, and thus a decision must be made to only treat the visible portion of the processes or (more commonly) to concurrently surgically remove the lens as in phacoemulsification cataract surgery, increasing cost.

FIGURE 5

↘ Intraoperative image of diode laser endoscopic cyclophotocoagulation, showing the ciliary processes undergoing laser therapy.
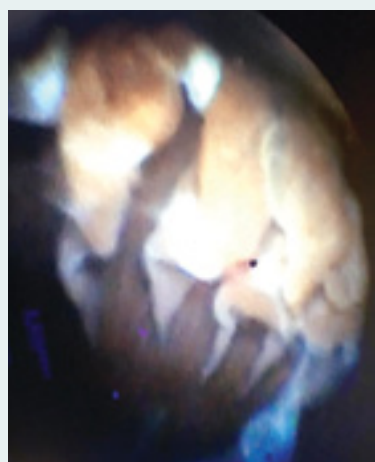
A video of the ECP procedure is available with this article and can be viewed here: https://www.mspca.org/wp-content/uploads/2018/03/ECP-1.mov. The video shows the ciliary processes being laser ablated from the bulbous head at the top of the screen to the tapering tail lower down. The red laser-aiming beam is visible, and the ciliary process turns white and contracts as it is lasered.

To the author's knowledge, there are no peer-reviewed publications on the use of ECP in dogs. However, in 2013 Lutz et al presented an abstract at the annual meeting of the American College of Veterinary Ophthalmologists (ACVO), reporting the outcomes in 309 dogs

TABLE 2

↘ Outcome of diode laser endoscopic cyclophotocoagulation for control of primary and secondary glaucoma in dogs, as reported by Lutz et al (2013)

YEAR	6 MONTHS	1 YEAR	2 YEARS	3 YEARS	4 YEARS
IOP Control	214/250 (86%)	190/230 (83%)	105/135 (78%)	37/61 (61%)	13/20 (65%)
Vision	192/229 (84%)	157/214 (73%)	83/123 (67%)	32/59 (54%)	7/16 (44%)

treated with ECP. Primary glaucoma was present in 86 dogs (97 eyes), and secondary glaucoma was present in 171 dogs (212 eyes). The outcomes for IOP control and vision in both of these cohorts combined are presented in Table 2.

THE FUTURE?

Finally, the newest reported modality available to treat canine glaucoma is the MicroPulse® system by IRIDEX Corporation. Returning to the transscleral method of energy delivery, this laser has an on-off duty cycle that allows the treated tissue to cool between bursts of energy. The spread of heat to adjacent tissues is thus reduced, decreasing the potential for complications.

MicroPulse® transscleral diode laser cyclophotocoagulation (mTSCPC) was reported in two abstracts at the 2017 ACVO conference. Sapienza et al reported a 53% (17/32) success rate for a single lasering and 75% (9/12) for repeat lasering, giving an overall 81% (26/32) success rate in controlling IOP. Sebbag et al controlled the glaucoma of 50% (7/14) of their cases, with IOP reducing 50-87%. These preliminary results are encouraging, but peer-reviewed studies with greater numbers of cases are needed, and to the author's knowledge, there are few places that have this laser available at the present time.

CONCLUSIONS

One-year outcomes are typically reported as a benchmark for success in glaucoma surgery, balancing the need for long-term data with loss of cases to follow up. The bulk of the data available in veterinary medicine on outcomes following glaucoma surgery are on transscleral cyclophotocoagulation, with or without Ahmed valve placement. Although not scientifically appropriate, combining all reported studies from 1997 to 2011 on TSCP shows one-year IOP control in 132/197 (67%) and one-year vision in 56/105 (53%). More peer-reviewed data are needed to better understand the benefits of endoscopic cyclophotocoagulation over TSCP. MicroPulse® therapy similarly awaits further investigation.

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↘ Counseling Support for Pet Parents

Angell's Annette Scanlon, LICSW is a veterinary licensed social worker (LICSW) and experienced with supporting individuals who are faced with making difficult veterinary medical decisions and dealing with grief and loss. Clients who face these challenges are often overwhelmed and unsure of what to do next. The veterinary social worker is skilled in assisting in these situations.

Clients referred to Angell are offered complimentary services to help them cope. Sessions are available both individually and in a group setting. The client can meet with our social worker to process their options for maintaining their pet's quality of life, end-of-life decisions, and working through their grief and loss. The group sessions are offered both weekly and monthly. Groups are provided for those in a caregiving role, those with support animals, and those who have experienced a pet loss.

Angell staff are also supported by the social worker. Support is provided for Angell veterinarians, veterinary technicians, and support staff to effectively handle ethical dilemmas, work with challenging clients, and learn how to identify and deal with compassion fatigue and burnout. angell.org/grief
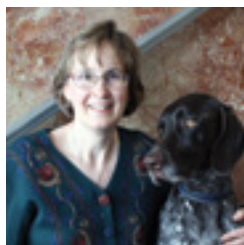
Color Atlas of Canine Cutaneous Round Cell Tumors

Patty Ewing, DVM, MS, DACVP (anatomic and clinical pathology)

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INTRODUCTION

Cytologic evaluation of cutaneous and subcutaneous mass aspirates is a convenient, rapid way of obtaining a definitive or presumptive diagnosis in a majority of patients. In one study comparing cytologic and histopathologic test results of cutaneous and subcutaneous lesions in 243 specimens, the diagnosis was in agreement in 90.9% of cases.[1] The results of cytologic evaluation may provide information about prognosis and guidance for next diagnostic steps and/or treatment. Sedation or anesthesia is rarely needed for sample collection. Samples can often be collected, prepared, and evaluated microscopically in a matter of minutes. The category of discrete round cell tumors is composed primarily of cells of the hemolymphatic system. In addition to the round to oval shape of the cells, the distinguishing morphologic feature is their discrete nature, which lacks cellular junctions, resulting in singly occurring cells rather than cohesive aggregates (epithelial tumors) or loose aggregates of cells associated with extracellular matrix (mesenchymal/spindle cell tumors).[2] Aspirates of round cell tumors generally yield a large number of neoplastic cells, making them rewarding to evaluate. In this color atlas, cytologic characteristics of the following six canine round cell tumors will be highlighted: canine cutaneous histiocytoma, histiocytic sarcoma, mast cell tumor, lymphoma, plasma cell tumor, and transmissible venereal tumor (TVT). Table 1 provides a summary of cytologic features and typical locations for each round cell tumor type.

TABLE 1

↘ Cytologic Characteristics of Cutaneous/Subcutaneous Discrete Round Cell Tumors

TYPE	COMMON APPEARANCE	CYTOLOGIC CHARACTERISTICS[2]
Histiocytoma	Single smooth, pin-raised hairless mass; often on head, pinnae	Relatively uniform, medium round cells with moderately sized round to slightly indented nucleus; finely etched chromatin; and a moderate amount of pale, slightly granular cytoplasm; few mitotic figures
Histiocytic sarcoma	Single or multiple purple/red nodules; often with visceral involvement or periarticular location	Large round to spindle cells with one or more large round nuclei, marked pleomorphism, and abundant pale cytoplasm often vacuolated or exhibiting phagocytosis of RBCs or WBCs; mitotic figures may be present
Mast cell tumor	Single or less often multiple white to light yellow or hemorrhagic masses or plaques; ulceration common; visceral involvement possible	Medium round cells with central round nucleus often obscured by numerous fine to coarse purple cytoplasmic granules filling moderately abundant pale cytoplasm
Lymphoma	Multiple off-white or red to purple nodules in nonepitheliotropic type	Medium to large round cells with high nucleus-to-cytoplasm (N:C) ratio, finely granular chromatin, prominent nucleoli, scant medium blue cytoplasm; mitotic figures common in high-grade lymphoma
Plasma cell tumor	Single raised pink nodule on head, trunk or limbs; usually not associated w/multiple myeloma	Medium round cells with a large round eccentric nucleus, coarsely reticulated to stippled chromatin, + indistinct nucleoli, and abundant medium to deep blue cytoplasm often with perinuclear clear zone; mitotic figures may be present
Transmissible venereal tumor	Single or more often multiple nodular pedunculated to cauliflower-like masses on external genitalia of sexually active dogs	Large round cells with moderately sized, round nuclei; coarsely stippled chromatin; small to prominent nucleoli; and a moderate amount of pale cytoplasm with few small distinct cytoplasmic vacuoles; mitotic figures may be present

PATHOLOGY

CONTINUED FROM PAGE 10

CANINE CUTANEOUS HISTIOCYTOMA

Histiocytomas are benign tumors composed of Langerhans cells, which are dendritic, antigen-presenting cells of the skin. Histiocytoma occurs most commonly as a solitary skin mass in dogs under two years of age but can occur in dogs of any age.[3] Scottish terriers, bull terriers, boxers, English cocker spaniels, flat-coated retrievers, Doberman pinschers, and Shetland sheepdogs are predisposed toward development of histiocytomas.[4] The majority of tumors undergo spontaneous regression due to immunologic reactivity.[4] Cytologic appearance is shown in Figure 1.

HISTIOCYTIC SARCOMA (MALIGNANT HISTIOCYTOSIS)

Histiocytic sarcoma may occur as localized masses in the subcutaneous tissue or periarticular region or as disseminated disease (lymph nodes, bone marrow, spleen, liver, lung, skin). The disseminated form is commonly referred to as malignant histiocytosis. Localized histiocytic sarcomas occur most commonly in middle-aged or older flat-coated retrievers, golden and Labrador retrievers, and rottweilers.[5] Malignant histiocytosis has a predilection for Bernese mountain dogs as well as the aforementioned breeds.[5] The disease is uniformly fatal after a progressive disease course. Cytologic appearance is shown in Figure 2.

FIGURE 1

↘ **Aspirate from a cutaneous histiocytoma. Left.** Note large, discrete round to oval cells (black arrows) of relatively uniform size that have a large, round to oval, slightly eccentric nucleus and moderate amount of cytoplasm. Low numbers of small lymphocytes (green arrow heads) and a plasma cell (red arrow head) are present. (Diff-Quik, 600x magnification.) **Right.** Higher magnification of histiocytoma cells showing finely etched chromatin with one to three small, indistinct nucleoli in the nucleus and abundant, slightly granular, pale blue cytoplasm. (Diff-Quik, 1500x magnification.)
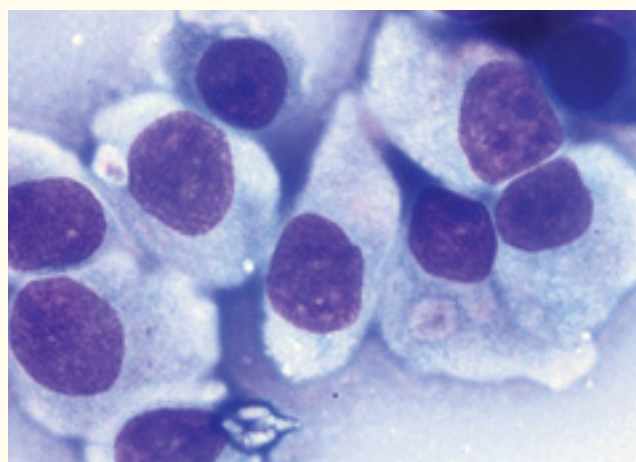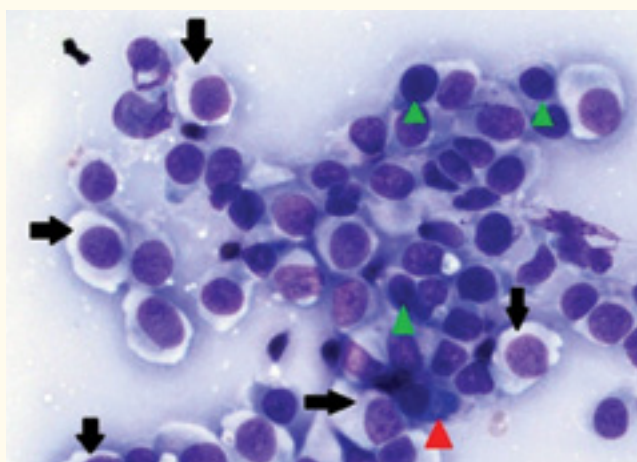
FIGURE 2

↘ **Aspirates of histiocytic sarcoma. Left.** Note large, discrete, singly occurring round to oval cells that have a large, round, eccentric nucleus; coarse chromatin; multiple round nucleoli; and moderate amount of vacuolated cytoplasm containing darkly stained hemosiderin pigment and phagocytized erythrocytes or leukocytes (black arrows). The green arrow identifies a neoplastic cell with a bizarre mitotic figure. Red arrow heads identify neutrophils for size comparison. (Diff-Quik, 1000x magnification.) **Right.** Histiocytic sarcoma with both round and pyriform to spindle-shaped cells (black arrows) arranged in noncohesive aggregates. Note multinucleation of some of the cells. (Diff-Quik, 500x magnification.)
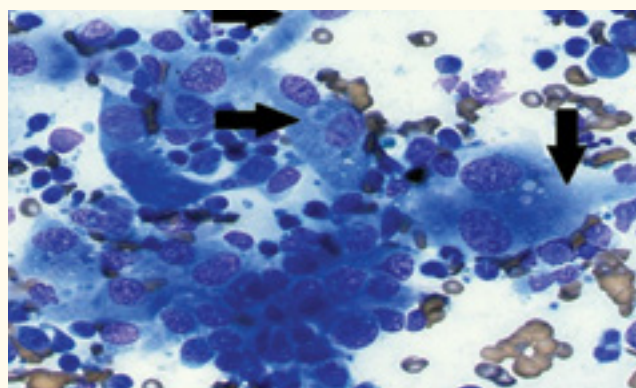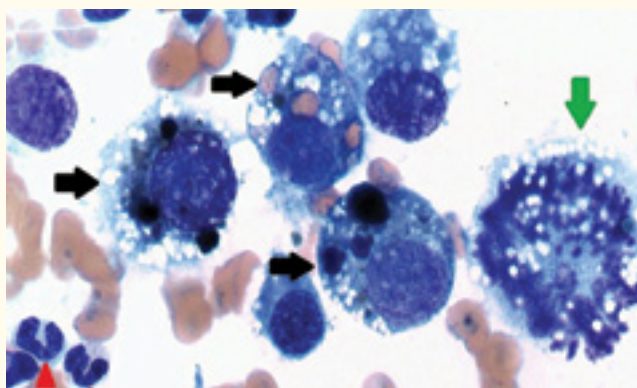
MAST CELL TUMOR

Mast cell tumors are one of the most rewarding round cell tumors to diagnose because they are readily identified by the presence of their distinctive purple mast cell tumors. Multiple dog breeds are predisposed to developing mast cell tumors, which may be solitary or multicentric. Most tumors occur in middle-aged and older dogs. A cytologic grading scheme for mast cell tumors was recently proposed and may be helpful in differentiating low-grade and high-grade tumors, although histologic evaluation remains the gold standard for mast cell tumor grading. In the study that evaluated this grading scheme, dogs with low-grade tumors had a prolonged survival relative to dogs with high-grade tumors (mean survival time of 364.6 + 42.5 days with high-grade tumors). Dogs with cytologic high-grade mast cell tumors were 25 times more likely to die within the two-year follow-up period than dogs with low-grade tumors. The grading scheme had 88% sensitivity and 94% specificity relative to the two-tiered histology grading system.[6] Cytologic appearance of a low-grade versus a high-grade tumor is shown in Figure 3.

LYMPHOMA

Cutaneous lymphoma exists in two different forms: 1) epitheliotropic, which is typically small to intermediate cell size, and 2) nonepitheliotropic, which is typical of intermediate to large cell size. Epitheliotropic lymphoma will not be covered here since this entity requires histopathology to identify epitheliotropism for making the diagnosis. Briards, English cocker spaniels, bulldogs, boxers, Scottish terriers, and golden retrievers are predisposed to cutaneous lymphoma.[5] Dogs with nonepitheliotropic lymphoma have single or more often multiple cutaneous nodules. The disease is progressive, with eventual involvement of lymph nodes and/or viscera. Cytologic appearance of nonepitheliotropic lymphoma compared to cutaneous plasmacytoma is shown in Figure 4.

PLASMACYTOMA (PLASMA CELL TUMOR)

Plasmacytomas typically arise de novo from cutaneous plasma cells and are not associated with bone marrow involvement. Multiple myeloma (plasma cell neoplasia in the bone marrow and other organs) can be associated with skin involvement, but this occurs very infrequently.[4] Older dogs are more commonly affected by cutaneous plasmacytomas. Cocker spaniels, Airedale terriers, Kerry blue terriers, standard poodles, Yorkshire terriers, and Scottish terriers are predisposed.[5] Most cutaneous plasmacytomas are cured with complete surgical excision. Cytologic appearance of plasmacytoma compared to lymphoma is shown in Figure 4.

TRANSMISSIBLE VENEREAL TUMOR (TVT)

The cell of origin of TVT remains a mystery, and it is unique among the round cell tumors in being transmitted by physical transplantation in sexually active dogs. The chromosome count is 59 rather than the normal 78 found in other cell types.[4] In addition to external genitalia, masses may be found on the lips and other portions of the skin or mucosa that come into contact with the genitalia. Tumors initially grow rapidly, then become static for a time and may eventually undergo spontaneous remission several months later. TVT infrequently metastasizes to regional lymph nodes and, rarely, to viscera.[4] Treatment with vincristine is highly effective at achieving complete remission in most dogs. Cytologic appearance of TVT is shown in Figure 5.

SUMMARY

Cytologic examination of cutaneous and subcutaneous round cell tumors is often rewarding due to the high cell yield on fine-needle aspiration. With practice, differentiation of the six distinct types of cutaneous round cells shown here can be readily achieved in the vast majority of cases. The results of cytologic evaluation may provide information about prognosis, guide next diagnostic steps, or allow for rapid treatment.

FIGURE 3

↘ **Aspirate of low-grade (left) and high-grade (right) mast cell tumors**. **Left.** Low-grade mast cell tumor has medium, discrete, round to oval cells (black arrows) of relatively uniform size with a medium, round central nucleus partially observed by numerous small purple granules that fill the abundant cytoplasm. (Diff-Quik, 1000x magnification.) **Right.** High-grade mast cell tumor has sparsely granulated, discrete, round cells exhibiting multinucleation (black arrows), bizarre mitotic figure (yellow arrow), and nuclear atypia featuring multiple prominent nucleoli. Green arrows identify neutrophils and eosinophils for size comparison. (Diff-Quik, 600x magnification.)
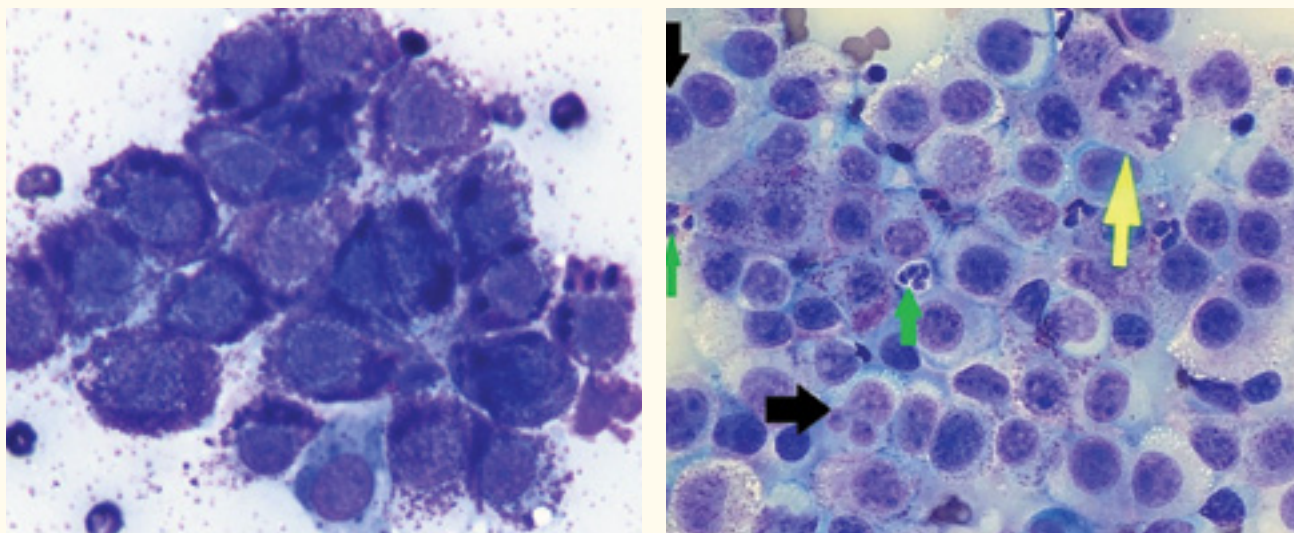
FIGURE 4

↘ **Cytologic appearance of lymphoma (left) compared to plasmacytoma (right). Left.** Note lymphoma cells are medium to large and have a higher N:C ratio and more finely stippled chromatin than the plasmacytoma. Yellow arrows identify non-neoplastic, small lymphocytes for size comparison. The green arrow identifies a bizarre mitotic figure. (Diff-Quik, 750x magnification.) **Right.** Neoplastic plasma cells have increased amounts of deep blue cytoplasm compared to lymphoma, with a more eccentrically placed nucleus that has a coarser or reticular chromatin pattern. Binucleation (black arrow) or multinucleation and prominent anisokaryosis (green arrows identify neoplastic cells with larger nuclei) are common features. Both lymphoma and plasmacytoma may display perinuclear clear zones and mitotic figures. (Diff-Quik, 750x magnification.)
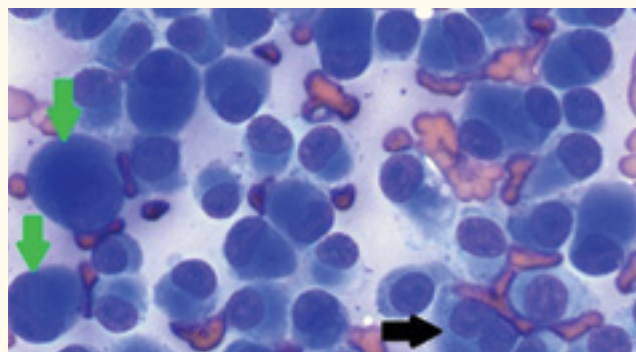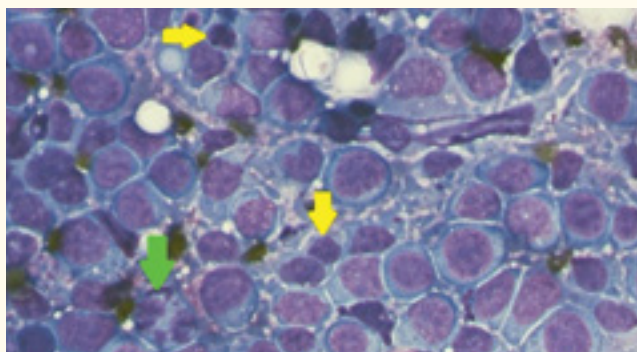

FIGURE 5

↘ **Cytologic appearance of transmissible venereal tumor. Left.** Note large, discrete, round cells with moderately sized, round, slightly eccentric nuclei and a moderate amount of pale blue cytoplasm. Green arrows identify neutrophils for size comparison. (Diff-Quik, 600x magnification.) **Right.** Higher magnification of neoplastic cells showing coarsely stippled chromatin, one or more small to medium-sized dark nucleoli, cytoplasmic vacuoles (thin red arrow), and a mitotic figure (black arrow). (Diff-Quik, 1200x magnification.)
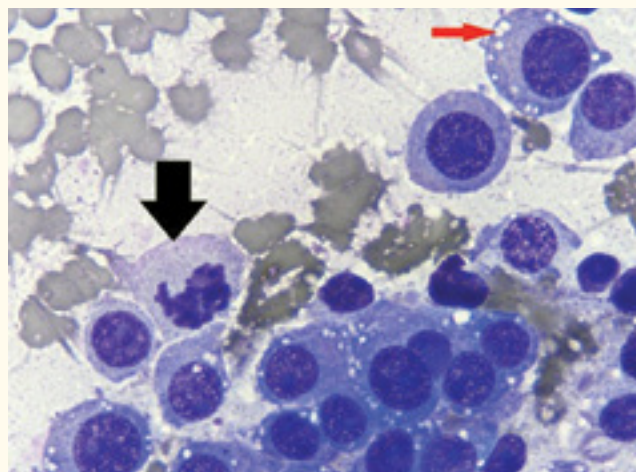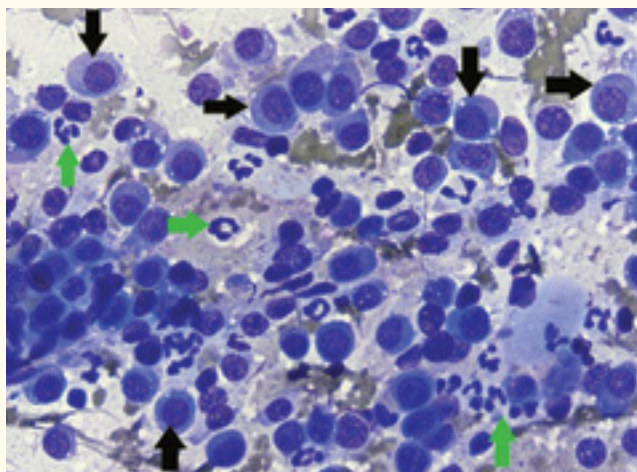
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Renal Replacement Therapy in Veterinary Medicine

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Renal replacement therapies (RRT) were first reported in companion animal veterinary medicine in the early 1990s. Although initially limited to a few specialty centers, these therapies are becoming more available throughout the United States as well as other countries around the world. Most often RRT is considered for acute kidney injury, but other applications include treatment of chronic kidney disease, acute on chronic kidney disease and exposure to various drugs and toxins.

RRT primarily relies on three basic principles for removal of uremic toxins. In all instances, a semipermeable membrane filled with thousands of small straws is used to separate patient blood from dialysate. Blood typically passes through these straws while dialysate passes on the outside. With blood running countercurrent to dialysate, small molecular weight molecules, typically < 500 Daltons, will move from the higher concentration in the blood to the dialysate via diffusion. Similarly, certain substances, such as bicarbonate, can pass from dialysate to the blood. By applying pressure across the membrane, water can be pushed through the membrane pores (ultrafiltration) and with water, small and middle-size molecules will follow through a process called solvent drag or convection. Balanced electrolyte replacement fluids are administered in this instance to avoid volume depletion. Adsorption contributes the least to removal of molecules but is the process by which molecules will adhere to the membrane.

Traditional intermittent hemodialysis (IHD) therapies use large amounts of dialysate that are generated from a purified water system. High blood and dialysate flows can be achieved with this, allowing diffusion to be the primary method of clearance. Intermittent hemodialysis can achieve fairly large clearances of major uremic toxins in short periods of time due to the faster processing of blood and dialysate. Conventional continuous renal replacement therapy (CRRT) is based on convective therapies, but CRRT machines, such as the Prismaflex®, also allow for diffusive clearance or a combination of both diffusive and convective clearance

(hemodiafiltration). In the past, machines used for IHD were used primarily for short (< 6 hours) and efficient treatments while continuous therapy provided a slower, more physiologic resolution to azotemia given for about 24 hours per day. However, systems and/or treatment plans have been adapted in recent years so that slow treatments or treatments for smaller patients can be achieved on IHD machines. Likewise, prolonged intermittent treatments (PIRRT) on CRRT machines closely resemble treatments on IHD machines. Additionally, there are a variety of dialyzer options available, depending on the machine type, that can alter treatment based on pore size, membrane surface area and ultrafiltration abilities.

Overhydration has been shown to be an independent predictor of death in people (CRRT vet clinics), and the human literature documents the negative effect of aggressive fluid therapy on survival, length of hospital stays, and oxygenation status. Clinical overhydration is common in veterinary patients, especially in those presenting in oliguric or anuric renal failure.[1,2] This is often due to attempts to convert patients to polyuria with aggressive fluid therapy. Ultrafiltration, achieved through the convection principle, can be used to only remove excess plasma water in cases of volume overload as well as refractory congestive heart failure. The percentage of overhydration is determined prior to therapy, and machines are set to remove the indicated volume slowly over the course of treatment. Care must be taken with fluid removal to monitor for hypovolemia as this amount of fluid is not being returned to the patient and rapid fluid removal may result in marked hemodynamic changes. An in-line monitor (Critline®) is used to help monitor these volume changes.

INTERNAL MEDICINE

Indications for dialytic intervention include significant or rising azotemia and oliguria or anuria in the face of appropriate medical management. Significant electrolyte abnormalities along with metabolic acidosis will respond to dialysis but are rarely indications for therapy by themselves. What constitutes significant azotemia and timing for intervention is still variable. In the literature, the majority of patients undergoing hemodialysis were reported to have a creatinine > 10mg/dL,[1,2,3,4] however, with severe azotemia comes increased risk of extra-renal uremic complications that are not always reversible with treatment. The decision for intervention should be considered on a case-by-case basis, but it is speculated that earlier interventions than what is in past reports may improve the survival statistics over time.

There are several complications that can occur in the intra- and interdialytic period. Patients undergoing therapy must be anticoagulated during the treatment.[5] If systemic heparinization is performed, patients may be at risk for hemorrhage even after the conclusion of therapy. Regional anticoagulation can be performed using citrate, which forms complexes with calcium, an important cofactor in the coagulation cascade. This form of anticoagulation can be associated with clinical hypo or hypercalcemia.[6] With both forms of anticoagulation, clotting can occur during treatment which may necessitate stopping treatment prematurely and possible administration of blood products due to blood loss in the line. Other common but often correctable complications include hypotension[1,2,3,8,9] and hypothermia[7]. Dialysis disequilibrium syndrome (DDS) is one of the most serious complications of dialysis therapy. Though the exact pathogenesis is not fully understood, clinical signs are thought to be due to rapid, dialysis-induced changes in the composition of blood. An osmotic concentration gradient leads to intracellular swelling and subsequent cerebral edema. Treatments over a longer period of time and/or smaller reductions in urea improvement for those that are significantly azotemia can reduce the risk of DDS.

The survival rates of dogs and cats undergoing hemodialysis are about 50%, which is similar to overall survival rates in patients with acute kidney injury.[4, 8, 9, 10, 11] A recent meta-analysis review looked at differences in survival between patients treated conservatively versus with hemodialysis, and although the mortality rate was higher in the dialysis group, the difference was not significant between groups.[4] In addition, the patients undergoing dialysis had more severe disease, including a higher rate of oliguria or anuria in comparison to the conservative group (88% versus 11.6%). Definition of survival across studies also makes it difficult to compare outcomes for patients, but in one study looking at long-term survival (> 365 days), ~35% of patients were still alive.[10] Besides severity of disease, etiology of the renal injury plays an important role in prognosis. Infectious (leptospirosis, pyelonephritis) and obstructive (ureterolith; cats) etiologies consistently have a better prognosis (~70-80% survival) even in the face of low urine output, compared to toxic (ethylene glycol) or ischemic causes.[1,2,8,9,10,11] Owners should be aware that even with treatment, some patients will have chronic kidney disease that may require medical management after the cessation of dialytic therapies.

At Angell Animal Medical Center, we are now pleased to be able to offer hemodialysis to our veterinary patients in addition to total plasma exchange. For more information, please feel free to contact either Dr. Shawn Kearns (skearns@angell.org) or Dr. Courtney Peck (cpeck@angell.org) For after-hour cases, please contact our Emergency department staff, who will then be in touch with us.

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Anaplasmosis
(*Anaplasma phagocytophilum*)

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Granulocytic anaplasmosis is a common tick-borne disease caused by a bacterial agent *Anaplasma phagocytophilum*. This disease is regularly found in the Northeast but is also found in the Midwest and along the West Coast. The disease is carried and spread by deer ticks, which are the same ticks that carry and spread Lyme disease. Therefore, Anaplasma and Lyme disease are commonly diagnosed together as coinfections.

A minimum tick feeding time of 24 hours is needed for the deer tick to transmit the infectious bacteria. The organisms infect white blood cells and can be detected anywhere from 4 to 14 days later. Therefore, the clinical signs are seen approximately one to two weeks after the tick bite. Aside from it affecting a particular white blood cell, it can cause a low platelet count. Though it is not known exactly how the platelets get affected, it can be a clue to the veterinarian of what is going on with the pet.

Many dogs exposed to *A. phagocytophilum* do not become infected and therefore do not develop obvious clinical signs. Those that do become infected can develop a fever, lethargy, inappetence, and general malaise. Some dogs may show lameness/stiffness, with swollen joints and big lymph nodes noted on physical exam. Less common clinical signs include vomiting, diarrhea, coughing, and respiratory difficulties. Because of the potentially low platelet numbers, signs of bleeding can occur, but this is not common. Your pet could have a nose bleed, small pink dots on the skin or gums, or bleeding within the intestines. Rarely, Anaplasma can affect the brain and cause a pet to have an unsteady gait and seizures.

Diagnosis of an Anaplasma infection may require several different laboratory tests, including bloodwork, a blood smear, or a 4DX test (test for heartworm and tick-borne diseases). Anaplasma and other tick-borne diseases are treated with antibiotics. The most effective antibiotic is doxycycline, but other antibiotics can be effective. This general class of antibiotics can be very harsh on the stomach and can cause inappetence, vomiting, or diarrhea. Anaplasma should be treated for a minimum of 14 days, but other tick-borne diseases may require longer treatment. Dogs that have a dangerously low platelet count as a result of infection may require a brief hospitalization for monitoring and restriction while treatment is initiated.
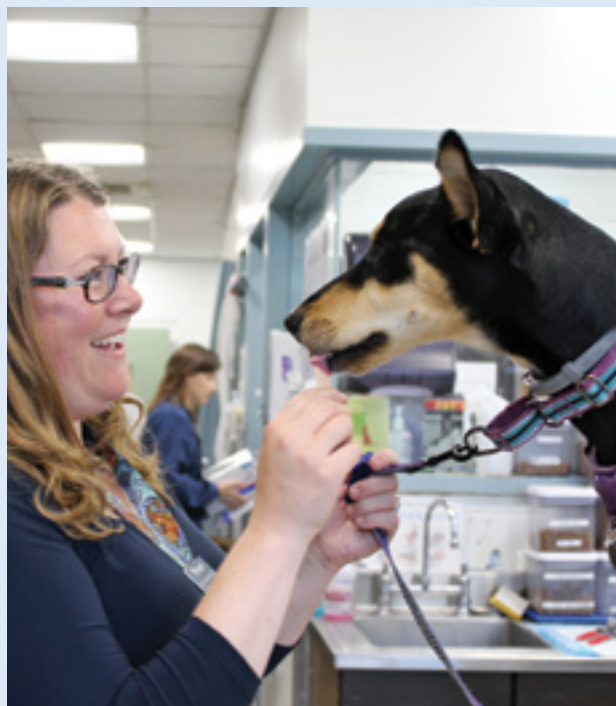
↘ Angell's Referring Vet Portal

angell.org/vetportal

24/7 access to your referred patients' records

We are pleased to offer the Angell Referring Veterinarian Portal to our referring partners. The Portal provides 24/7, secure, mobile-friendly access to your referred patients' records through **angell.org/vetportal**. The system automatically updates throughout the day and provides 24/7 access to:

- Online Medical Records
- SOAPs
- Lab Results
- Discharge Instructions
- Check-in Status
- Diagnostic Images
- Referral Reports
- Prescriptions

Settings can be customized within the Portal to receive notices by email or fax, and you may list multiple emails to receive check-in, discharge, deceased, and update notices.

Visit **angell.org/vetportal** or call our referral coordinator at **617 522-5011** to gain access to your account.

↘ Physical Rehabilitation at MSPCA-Angell West

Canine physical rehabilitation is used to treat a wide variety of orthopedic and neurological conditions. Whether recovering from an injury, cross training, or facing mobility issues, dogs can substantially benefit from physical rehabilitation.

MSPCA-Angell West Physical Rehabilitation offers services seven days per week. Jennifer Palmer, DVM, Certified Canine Rehabilitation Therapist (CCRT), and Amy Straut, DVM, CCRT, lead our Physical Rehabilitation team. Visit **angell.org/rehab** for details and video footage of the impact of their work on our canine patients.

Current physical rehabilitation services include:

- Hydrotherapy
- Manual therapy
- Massage
- Land-based exercise
- Therapeutic laser treatment
- Consultation and fitting of assistive devices
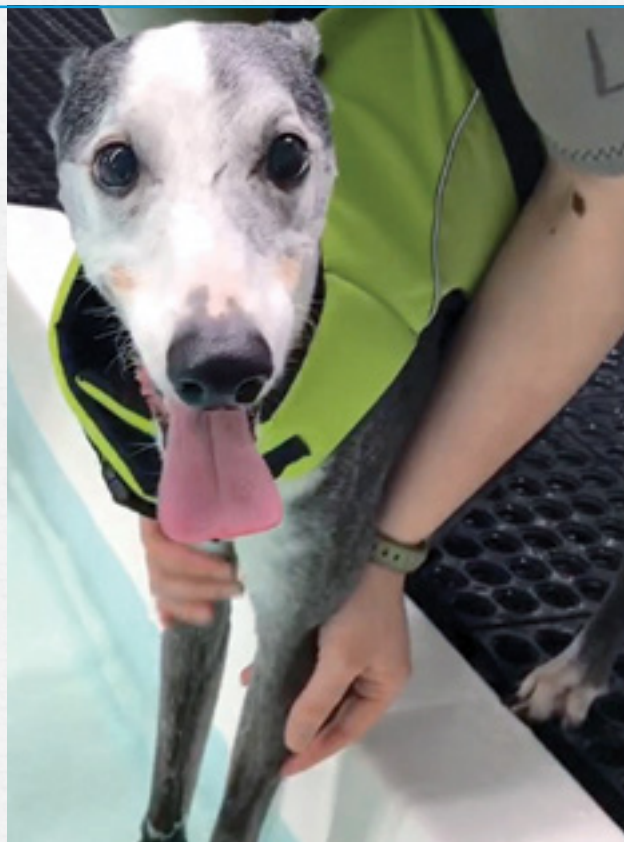
↘ Angell At Nashoba: Low-Cost Care for Financially Qualified Clients

Angell Animal Medical Center and Nashoba Valley Technical High School operate **Angell at Nashoba**, a veterinary clinic for low-income pet owners that also serves as a rigorous academic and experiential training program for students enrolled at Nashoba Valley Technical High School.

The clinic provides discounted:

- Primary Veterinary Care
- Surgery and Dental Services

Open weekdays from 7:45 am-4:00 pm throughout the year, the clinic does not provide overnight care, specialty service care, nor 24/7 emergency service as Angell's Boston and Waltham facilities do, but will refer cases as appropriate to surrounding specialty veterinary referral hospitals.

To reach the clinic, please call **978 577-5992**. The clinic is located at 100 Littleton Road, Westford, Massachusetts.

For more information, visit **angell.org/nashoba**.

↘ Dr. Laurence Sawyer, Medical Director of Angell at Nashoba.

STAFF DOCTORS AND RESIDENTS

■ We encourage you to contact Angell's specialists with questions.

Main Phone: 617 522-7282 (Boston) | 781 902-8400 (Waltham) | Veterinary Referrals: 617 522-5011 | Angell at Nashoba: 978 577-5992

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(W/B) Services also available at our Waltham location

⬂ Our Service Locations

BOSTON & WALTHAM	BOSTON ONLY
Avian & Exotic Medicine 617 989-1561	**Anesthesiology** 617 541-5048
Behavior 617 989-1520	**Dentistry** 617 522-7282
Cardiology 617 541-5038	**Dermatology** 617 524-5733
Diagnostic Imaging 617 541-5139	**Nutrition** 617 522-5011
Internal Medicine 617 541-5186	**Oncology** 617 541-5136
Neurology 617 541-5140	**Ophthalmology** 617 541-5095
Physical Rehabilitation** 781 902-8400	**Pain & Palliative Care** 617 541-5140
Surgery 617 541-5048	**Pathology*** 614 541-5014

24/7 Emergency & Critical Care
■ Boston: 617 522-5011   ■ Waltham: 781 902-8400

*Boston-based pathologists serve both Boston and Waltham locations **Available only in Waltham

angell
Kindness and Care for Animals®

We mail one complimentary copy of our newsletter to each of our referring partners. Please circulate this copy within your practice.

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Please consider adding Angell's main numbers to your after-hours phone message.

■ angell.org/directions (free parking) ■ angell.org/hours ■ angell.org/ce

↘ Angell Expands Dentistry Service, Welcomes New Staff

The Dentistry Service at Angell Animal Medical Center is pleased to welcome Alice Ekerdt, DVM as the newest member of the Dentistry team.

Dr. Ekerdt joins Drs. Jessica Riehl, Erin Abrahams, and Colleen McCarthy. The team has doubled in size since the start of 2018 to meet the needs of patients, including those with high anesthesia risk.

617 522-7282 | dentistry@angell.org
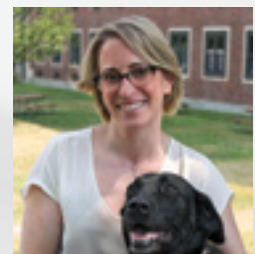angell.org/dentistry

Angell Welcomes New Staff

Dr. Alice Ekerdt was born and raised in Austin, Texas. She received her undergraduate degree from the University of Texas and her Doctorate of Veterinary Medicine from The Ohio State University. She worked in general practice for six years prior to starting her dentistry residency in New York. Dr. Ekerdt is a member of the Foundation for Veterinary Dentistry and the American Veterinary Medical Association.

Jessica Riehl, DVM, DAVDC

Erin Abrahams, DVM

Colleen McCarthy, DVM